907 Novel Uses of Contrast Echocardiography

Wednesday, April 1, 1998, 4:00 p.m.–5:00 p.m.
Georgia World Congress Center, Room 367W

4:00

907-1 The Impact of Stimulated Acoustic Emission on Myocardial Contrast Echocardiography – First Clinical Results

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Previous both in-vitro and animal studies have shown that micro-bubbles of Levovist® act as active soundsources during disintegration as they emit a broad band frequency spectrum (stimulated acoustic emission (SAE)). Due to the onset of SAE it is possible to receive Doppler-signals even from stationary bubbles or from bubbles with very low flow velocities. Hence, during their spectral characteristics they are beyond the wall filter frequencies and have a high signal amplitude. However, it is unknown whether SAE is applicable in humans to display myocardial contrast effects. Accordingly we evaluated myocardial contrast (MC) effects in 36 patients with coronary artery disease following intravenous bolusinjection of 3g Levovist. Harmonic Power Doppler investigations (H-PDI) were performed using a modified commercially available ultrasound machine (HDI 3000, ATL). Intermittent scanning was performed one frame every 5th cardiac cycle. MC was assessed using a four step visual score (0 = no contrast, 3 = complete MC). Contrast effects were evaluated using a calibratou software tool in both myocardium and cavities.

Results: MC score was 1.6 ± 0.6 using usual transmit Power (MI 0.8–1) and 2.7 ± 0.3 with MI = 1.5 ($p < 0.001$). Delineation of myocardial contrast from contrast signals of the cavities was possible while signal intensities in the cavities even at the endocardial border were at least 20 times higher than those of MC. In all registrations contrast signals within the LV-cavity appeared at least 5 cycles earlier than MC. MC appeared always first in basal segments of the myocardium.

Conclusion: H-PDI with high transmit power is useful for assessment of SAE. This approach improves MC following iv injection of Levovist.

4:15

907-2 A New Tissue Targeted Ultrasound Contrast Agent, MRX408 Improves Visualization and Delineation of Left Atrial Appendage Clot With Conventional 2-dimensional Echocardiography

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Background: Identification of LA appendage thrombus (LAAT) has important implications. Despite the utility of transesophageal echocardiography, reliable recognition of LAAT could be difficult. MRX408, made of lipid coated perflurobutane microbubbles, is a new ultrasound contrast that targets thrombus. We hypothesized that MRX408 would enhance LAAT signals and permit differentiation of clot from adjacent issue.

Methods: To assess this, we attempted to create in vivo LAAT in 8 dogs and imaged the heart with conventional 2-D echocardiography at baseline, during infusion of a nontargeted contrast, MRX113 and during *targeted* MRX408. LAAT area and Videointensity (VI) were blindly analyzed. Presence or absence of LAAT was verified postmortem.

Results: (M ± SD): In 2 of 8 dogs, there was no contrast enhancement of LAA zone and autopsy showed no clot. In the other 6 dogs, there was visually apparent contrast enhancement in LAA zone with MRX408 but not with MRX113. Autopsy showed LAAT in all 6. VI of LAAT in these dogs was 53.5 ± 13.9 at baseline, slightly higher with MRX113 (79.7 ± 29.5) but was much higher with MRX408 (93.9 ± 34.4, $p < 0.01$ vs baseline, $p < 0.04$ vs MRX113). The full extent of LAAT (area, cm2) was evident with MRX408 (7.40 ± 4.31) when Compared to baseline (3.60 ± 2.83, $p < 0.02$) and MRX113 (4.06 ± 2.72, $p < 0.02$).

Conclusion: MRX408, a thrombus targeting contrast agent, enhances the echointensity of LAA clot and demonstrates its extent. It is a promising agent for the clinical detection of LAA clot.

4:30

907-3 Direct *in vivo* Recordings of Cavitational Activity Within the Anterior Myocardium During Intermittent and Conventional Harmonic Imaging Following Intravenous Ultrasound Contrast

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Background: In vitro studies have demonstrated that the peak negative pressures (PNP) used for diagnostic ultrasound imaging can produce cavitation of perfluorocarbon filled microbubbles, but *in vivo* demonstration of this has never been observed.

Methods: We measured root mean square cavitational activity (CA) with a 20 MHZ single crystal transducer aligned so that its focal region was within the anterior myocardium of two open chest dogs. The heart was simultaneously imaged with a 1.7 megahertz harmonic transducer using PNP of 0.3–0.9 megapascals (MPa) and frame rates (FR) of 43 Hertz (Hz) 10 Hz, and one FR every one (1:1) to three (1:3) cardiac cycles (CC). Background subtracted anterior myocardial videointensity (MVI) was measured off-line. Intravenous (IV) perfluorocarbon exposed microbubbles (PCB) were given as either a bolus (0.0025 ml/kg) or continuous infusion (CI).

Results: Results are shown (*$p < 0.05$ compared to all other groups):

	0.5–0.9 MPa			
	43 Hz	10 Hz	1:1	1:3
CA (mV)	1.6 ± 0.2	1.7 ± 0.2	2.1 ± 0.4	2.7 ± 0.8
MVI (units)	15 ± 7	30 ± 14	57 ± 16	81 ± 20*

CA *increased* as FR *decreased*, reaching its maximum at the longest FR (1 every 3CC). MVI was directly proportional to CA at 0.9 MPa ($r = 0.93$, $p < 0.0001$) and 0.5 MPa ($r = 0.71$; $p < 0.001$), but did not correlate with CA when using 0.3 MPa.

Conclusion: In vivo cavitation of PCB occurs in response to diagnostic ultrasound pressures from commercially available transducers. Lower FR and higher PNP *increase* intramyocardial CA of intravenously injected PCB, and may be the mechanism for enhanced myocardial contrast with intermittent harmonic imaging.

4:45

907-4 Myocardial Contrast Echocardiography for the Detection of Coronary Artery Disease

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Background: The accuracy of intravenous (IV) infusion of a PESDA solution (sonicated mixture of 1 ml 20% serum albumin, 12 ml 5% dextrose and 8 ml decafluorobutane gas), associated to newer ultrasound imaging techniques to detect coronary artery disease (CAD) in humans, has not been evaluated in a large series of patients (pts). Accordingly, we assessed the ability of IV PESDA to produce myocardial contrast echocardiography (MCE) perfusion defects in pts submitted to coronary angiography (ANG) to investigate CAD.

Methods: In 92 pts (66 male, 62 ± 11 years) submitted to ANG, MCE with IV PESDA was obtained using different protocols with Second Harmonic (SH), Power Harmonic (PH) imaging and triggering mode (TM). ANG was normal in 15 pts (Group I), with residual lesion <50% after reperfusion in 15 pts (Group II) and with obstruction >80% in 62 pts (Group III). To provoke a vasodilator response dipyridamole (33 pts) or adenosine (20 pts) were used. A homogeneous appearance of contrast enhancement distribution was established as normal perfusion pattern (NP), and absence or heterogeneous enhancement as a perfusion defect (PD).

Results: MCE showed NP in all except one pt in Group I and had a PD in 100% of pts in Group III (in 5 pts only after stress with diyridamole (3 pts) or adenosine (2 pts)). Group II had NP in 9 pts and a PD was detected in 6 pts.

Conclusion: A PD observed with MCE at rest or after stress strongly correlates with the presence of CAD, and may be used to detect CAD in humans.